

# Transcatheter closure of atrial septal defects in adults with the Amplatzer septal occluder

R Dhillon, B Thanopoulos, G Tsaousis, F Triposkiadis, M Kyriakidis, A Redington

## Abstract

**Objective**—To assess the efficacy and complications of device occlusion of atrial septal defects in adults, using the Amplatzer septal occluder (ASO).

**Design**—A prospective interventional study.

**Setting**—Paediatric cardiology departments in two European teaching hospitals.

**Patients**—The first 20 patients accepted for atrial septal defect device occlusion, on the basis of transoesophageal echocardiography. Sixteen patients had larger defects with right heart dilatation, while the primary indication for closure in four was a history of early paradoxical embolism.

**Interventions**—Transcatheter atrial septal defect occlusions performed under transoesophageal echocardiography and fluoroscopic guidance between December 1996 and June 1998.

**Outcome measures**—Success of deployment of ASO devices, procedure and fluoroscopic times, complications, and symptoms.

**Results**—The ASO device was successfully implanted in all 20 patients (14 female), median age 44.2 years, with no complications. Of the 16 patients with right heart dilatation, the median Qp:Qs was 2.5:1. Defects measured 11–22 mm (median 18) on transoesophageal echocardiography, with balloon sized diameter (and device size) of 13–28 mm (median 20). For all 20 patients, the procedure time ranged from 38–78 minutes (median 61), and fluoroscopy 8.4–24.7 minutes (median 15.2). There were residual shunts in three patients at the end of the procedure, which were trivial ( $\leq 1$  mm) as assessed by transoesophageal echocardiography, and persisted for more than six months in only one patient. Follow up ranged from 0.1–1.5 years (median 0.7). There have been no late complications.

**Conclusions**—The ASO device can be used successfully to close selected oval fossa defects in adults, with minimal procedural morbidity and excellent early results.

(Heart 1999;82:559–562)

**Keywords:** atrial septal defect; interventional cardiac catheterisation; Amplatzer septal occluder

Atrial septal defects are common, accounting for 6–10% of congenital cardiac lesions.<sup>1</sup> The increased pulmonary blood flow which results may lead to right heart volume overload, and

ultimately to symptoms of congestive heart failure and arrhythmia. For these reasons, closure of the defect is now usually recommended during early childhood.

Surgical closure of atrial septal defects in adults has been the subject of much recent debate,<sup>2–5</sup> and the closure of patent foramen ovale in young adults with early stroke is even more controversial. Nonetheless, atrial septal defect remains the most common diagnosis in adults undergoing surgical repair of congenital heart defects.<sup>6</sup> Although the results of surgical closure in adults are good, and symptomatic improvement is common,<sup>7</sup> if the current trend towards transcatheter closure of atrial septal defect in children could be translated to adults, then many patients could avoid the need for open heart surgery.

Since the first description of atrial septal defect device occlusion at cardiac catheterisation in 1976,<sup>8</sup> a number of different devices have become available. For many institutions, the Amplatzer septal occluder (ASO) (AGA Corporation, Minnesota, USA), has become the device of choice for atrial septal defect occlusion in children. In this study, we examined our initial experience with the ASO in the closure of interatrial communications in adults.

## Patients and methods

### PATIENTS

This study reports the personal experience of a single operator in each of two centres, in the transcatheter ASO closure of atrial septal defect in 20 adults. Eleven defects were closed in one institution (Royal Brompton Hospital, London, UK), and another operator performed the other nine occlusions (Aghia Sophia Children's Hospital, Athens, Greece). The indications for closure were large defects resulting in right heart dilatation (16 patients), with coexisting symptoms of exercise intolerance (nine of 16 patients) or arrhythmia (five of 16 patients), or smaller defects in patients with early stroke and presumed paradoxical emboli (four patients). Two patients with right heart dilatation had also experienced paradoxical emboli. Only four patients were asymptomatic.

All patients underwent transoesophageal echocardiography before attempted transcatheter closure. Atrial septal defect closure was attempted for all defects with an unstretched diameter of  $< 25$  mm, which were sufficiently remote from the pulmonary veins, systemic veins, or atrioventricular valves. A small ( $< 2$  mm) collocated fenestration was not considered a contraindication, but those with larger defects or multiple fenestrations were excluded.

Department of  
Paediatric Cardiology,  
Royal Brompton and  
Harefield Hospital  
NHS Trust, London,  
UK

R Dhillon  
A Redington

Department of  
Paediatric Cardiology,  
Aghia Sophia  
Children's Hospital,  
Athens, Greece  
B Thanopoulos  
G Tsaousis  
F Triposkiadis  
M Kyriakidis

Correspondence to:  
Professor A Redington,  
Department of Paediatric  
Cardiology, Great Ormond  
Street Hospital Trust,  
London WC1N 3JH, UK.

Accepted for publication  
2 February 1999

Table 1 Patients and procedures: transcatheter atrial septal defect closure by ASO in 20 adults

Patients									Procedures			
Patient number	Sex	Age (years)	Follow up (years)	Symptoms		Anticoagulation		Qp:Qs	TOE diameter (mm)	Device size (mm)	Procedure time (mins)	Fluoroscopy (mins)
				Pre-catheter	Post-catheter	Pre-catheter	Post-catheter					
1	F	58.3	0.36	PE	None	W	A,H,D,W	1.2	8	13	65	14.3
2	F	50.8	0.47	PE	None	W	A,H,D,W	1.1	7	12	61	10.8
3	F	50.3	0.24	PE	None	A	A,H,D	2.0	8	9	69	12.8
4	M	45.2	0.89	Angina, PE, EI	Angina	W	A,H,W	1.2	12	14	62	9.6
5	F	40.8	0.38	P	Paraesthesia, P	None	A,H,D	2.5	18	24	58	24.7
6	F	33.9	0.24	EI	EI	None	A,H	1.3	11	13	48	8.4
7	F	28.1	0.72	EI	None	None	A,H	3.0	14	20	48	9.7
8	F	24.2	0.15	AVRT	Blurred vision	None	A,H,D	2.6	22	28	64	12.3
9	F	17.3	0.72	A	None	None	A,H	2.3	15	20	69	14.1
10	F	16.1	0.17	EI	None	None	A,H	3.3	12	20	61	12.4
11	M	21.2	0.10	PE	None	A	A,H,D	1.3	8	12	38	9.4
12	M	28.8	1.53	A	None	None	A	2.6	19	21	66	23
13	F	45.0	1.50	EI	None	None	A	2.3	17	18	78	23
14	F	59.8	1.53	Afl,PE	Afl	W	A,W	3.0	22	26	61	20.1
15	M	50.3	1.25	EI, PAC	None	None	A	2.5	18.5	22	63	23.7
16	F	53.9	1.23	EI	None	None	A	2.6	17.6	20	61	22.8
17	M	45.0	1.33	EI,PAC	None	None	A	2.4	18.1	24	51	16.5
18	M	43.4	1.00	A	None	None	A	2.4	19	20	67	16
19	F	37.7	0.96	EI	None	None	A	2.8	19	22	48	20.2
20	F	45.6	0.74	A	None	None	A	2.3	19	19	54	17.2

Patients 1–11 from London, 12–20 from Athens.

Symptoms: A, asymptomatic; PE, paradoxical embolism; EI, effort intolerance; P, palpitations; AVRT, atrioventricular re-entry tachycardia; Afl, atrial flutter; PAC, premature atrial contractions.

Anticoagulation: A, aspirin; W, warfarin; H, heparin infusion; D, dalteparin sodium (low molecular weight subcutaneous heparin).

Device size, balloon sized diameter.

This study describes the 20 patients (14 female) accepted on the basis of transoesophageal echocardiography, who underwent cardiac catheterisation between December 1996 and June 1998. They were aged 16.1–59.8 years (median 44.2), and have been followed up for 0.10–1.53 years (median 0.73). None of the patients was subsequently excluded, and therefore all 20 underwent device implantation. One patient had previously undergone cardiac catheterisation in March 1998, but device implantation had not been attempted because the stretched diameter of the defect was 28 mm; at that time, the largest device available was 26 mm.

#### METHODS

All procedures were performed under general anaesthesia, with transoesophageal echocardiography guidance throughout. Single plane fluoroscopy was used, and angiography was not routinely performed. Following confirmation of an anatomically suitable defect by transoesophageal echocardiography, a right heart catheterisation was performed via a single femoral venepuncture, which was later used to introduce the delivery sheath and ASO. Heparin, 100 units/kg, was administered intravenously early in the procedure. Balloon sizing was performed in the usual way for the 16 patients with large atrial septal defects, but in those with paradoxical emboli care was taken to size the defect additionally by passing the balloon from the right atrium to left atrium, the largest stretched diameter being taken. De-airing of the delivery sheath was performed with the sheath in the inferior caval vein or right atrium, which was then advanced into the left atrium over the guidewire. Furthermore, the device itself was vigorously flushed with saline before introduction, via a valved, Y shaped connector placed on the proximal end of the loading catheter. Deployment and

subsequent intraprocedural assessment was almost entirely by transoesophageal echocardiography.

Transthoracic echocardiography was performed between 3–16 hours following device implantation. Further follow up at three months and one year included transthoracic echocardiography, according to the manufacturer's investigational protocol.

#### Results

The anthropometric data and procedural details are given in table 1. Device implantation was successful in all 20 patients, with no early or late embolisation. Of the 16 patients with atrial septal defects > 10 mm diameter as assessed by transoesophageal echocardiography, the Qp:Qs ranged from 1.2:1 to 3.3:1 (median 2.5:1). Their defects ranged in size from 11–22 mm (median 18) on transoesophageal echocardiography, with balloon sized diameter (and therefore device size) of 13–28 mm (median 20). For the entire group of 20 patients, the procedure time was 38–78 minutes (median 61), and fluoroscopy time 8.4–24.7 minutes (median 15.2).

In all but three patients, there was complete occlusion of the atrial septal defect by the end of the procedure. The residual shunts in these three patients were trivial ( $\leq 1$  mm) as assessed by transoesophageal echocardiography. Their defects had measured 20–21 mm on balloon sizing. In one patient this shunt has persisted at one year follow up. In the other two patients there was complete occlusion within 24 hours and six months, respectively. All patients were discharged within 24 hours, with the exception of one patient with multiple previous thromboembolic events, who was positive for factor V Leiden. She was maintained on a heparin infusion for two days, while anticoagulation with warfarin reached a therapeutic level.

Although there were no complications related to device implantation, five patients reported symptoms following device occlusion. In three of them, these had been present before intervention (angina in the presence of coronary artery disease, intermittent atrial flutter, and breathlessness on exertion, respectively). New symptoms following intervention were "glove and stocking" paraesthesia two days after intervention in one (patient 5), and unilateral segmental blurred vision five days after device implantation in the other (patient 8). In both patients the symptoms lasted less than 24 hours. Clinical review by a neurologist revealed no physical abnormalities, and the symptoms in patient 8 were attributed to atypical migraine. In a further patient (patient 6), there was an unexplained deterioration of pre-existing breathlessness with effort intolerance, which improved gradually over the course of three months. All five patients underwent transthoracic echocardiography which confirmed satisfactory device positioning, no obvious thrombus formation, and good ventricular systolic function. Patient 6 also underwent a ventilation:perfusion scan, which was normal.

Although all patients received intravenous heparin, 100 units/kg, during the intervention, their subsequent anticoagulant regimen varied according to the centre in which the intervention was performed, and whether they were perceived as being at high risk. All 20 patients were prescribed aspirin in an antithrombotic dose (300 mg once daily) for at least three months.

Of the 11 Royal Brompton Hospital patients, all received an overnight heparin infusion (one for two days), and three received long term warfarin. Two with large atrial septal defects, and three of the six patients with paradoxical emboli, subsequently received subcutaneous low molecular weight heparin (dalteparin sodium) at a dose of 2500–5000 units once daily, for 5–21 days. Two patients with paradoxical emboli also continued with their preprocedural regimen of warfarin. There have been no postprocedural embolic symptoms in any patients.

### Discussion

Although this study reports the experience in a small group of selected patients, the initial results of ASO device occlusion of atrial septal defect in adults is encouraging. The device was successfully deployed in all 20 patients selected by preprocedural transoesophageal echocardiography. There were no procedural complications, and the mean procedure time of 1 hour should become even shorter with more experience. The physiological results were also satisfactory. There were trivial shunts at the end of the procedure (residual defect  $\leq 1$  mm) in three patients, but this has persisted in only one (at one year follow up), and is of no haemodynamic significance.

It is too early to be sure whether symptomatic benefit will be achieved in either the group with large atrial septal defects and right heart dilatation, or smaller defects with early

stroke. The rationale for surgical repair in the former group remains a subject of debate.<sup>2–5</sup> A recent study has shown improved survival and functional status in adults following surgical repair of atrial septal defect, as compared to those managed medically, without closure.<sup>9</sup> Our own surgical data suggest that clinical improvement can occur even in patients formerly describing themselves as asymptomatic.<sup>7</sup>

The closure of patent oval foramina or small atrial septal defects in patients with otherwise unexplained early stroke is even more contentious, and its utility even more difficult to prove. This study cannot in any way address this issue, other than showing that in highly selected patients, who otherwise would have been referred for surgery by their neurologists and physicians, adequate closure of the intra-cardiac lesion can be achieved using the ASO device. The procedure is performed in the same way as for larger atrial septal defects, with the exception of additional balloon sizing from right to left in case of a valve effect of the remnant of the flap of the oval fossa.

The postprocedural anticoagulant regimen reflected differing institutional preferences. In the Royal Brompton Hospital patients, because of concerns over extrinsic material implanted into the cardiac chambers, and enhanced thrombotic tendency in adults (most of whom were women), an overnight heparin infusion was administered in all cases, following a bolus during the procedure. In all patients who had been receiving warfarin before the procedure, because of paradoxical embolism, this was continued. In practice, those patients at the Royal Brompton Hospital over 35 years old received dalteparin sodium, warfarin, or both. Those under 35 years did not, with the exception of one patient with previous paradoxical embolism, and one patient with previously documented atrioventricular re-entry tachycardia.

The ASO device is particularly useful in adults with atrial septal defect. Defects of up to 34 mm can now be closed, and larger devices are in development. Not all defects are suitable or ideal, however. We excluded patients with multiple defects or fenestrations, preferring to use a non-self centring device in some of these patients.<sup>10</sup> A similar experience has recently been reported by others. Kaulitz *et al* recently reported successful closure of multiple and aneurysmal defects in six children and one adult using the Cardioseal device.<sup>11</sup> A variant of the ASO device, with a larger external to central diameter ratio, is also being developed to cope better with such defects.

In summary, the ASO device can be used successfully to close selected oval fossa defects in adults, with minimal procedural morbidity and excellent early results.

1 Feldt RH, Avasthey P, Yoshimasu F, *et al*. Incidence of congenital heart disease in children born to residents of Olmsted County, Minnesota, 1950–1969. *Mayo Clin Proc* 1971;46:794–9.

2 Oakley CM. Closure of atrial septal defect in adult life. *Cardiologia* 1996;41:31–4.

- 3 Shah D, Azhar M, Oakley CM, et al. Natural history of secundum atrial septal defect in adults after medical or surgical treatment: a historical prospective study. *Br Heart J* 1994;71:224-7.
- 4 Oakley CM. Does it matter if atrial septal defects are not diagnosed in childhood? *Arch Dis Child* 1996;75:96-9.
- 5 Perloff JK. Surgical closure of atrial septal defect in adults. *N Engl J Med* 1995;333:513-14.
- 6 Berdjis F, Brandl D, Uhlemann F, et al. Adults with congenital heart defects: clinical spectrum and surgical management. *Herz* 1996;21:330-6.
- 7 Gatzoulis MA, Redington AN, Somerville J, et al. Should atrial septal defects in adults be closed? *Ann Thorac Surg* 1996;61:657-9.
- 8 King TD, Thompson SL, Steiner C, et al. Secundum atrial septal defect: nonoperative closure during cardiac catheterization. *JAMA* 1976;235:2506-9.
- 9 Konstantinides S, Geibel A, Olschewski M, et al. A comparison of surgical and medical therapy for atrial septal defect in adults. *N Engl J Med* 1995;333:469-73.
- 10 Redington AN, Rigby ML. Transcatheter closure of interatrial communications with a modified umbrella device. *Br Heart J* 1994;72:372-7.
- 11 Kaulitz R, Paul T, Hausdorf G. Extending the limits of transcatheter closure of atrial septal defects with the double umbrella device (Cardioseal). *Heart* 1998;80:54-9.